JACC Vol. 6, No. 5
November 1985:995–1003

A Prospective Clinical, Scintigraphic, Angiographic and Functional Evaluation of Patients After Inferior Myocardial Infarction With and Without Right Ventricular Dysfunction

DAVID E. HAINES, MD, GEORGE A. BELLER, MD, FACC, DENNY D. WATSON, PHD, THOMAS W. NYGAARD, MD, FACC, GEORGE B. CRADDOCK, MD, FACC, ANN A. COOPER, RN, ROBERT S. GIBSON, MD

Charlottesville, Virginia

To elucidate the functional and prognostic significance of right ventricular dysfunction after acute inferior wall myocardial infarction, 74 consecutive patients with inferior infarction were prospectively evaluated with gated equilibrium blood pool imaging at rest, submaximal exercise thallium-201 scintigraphy and coronary angiography before hospital discharge. In addition, symptom-limited stress thallium-201 scintigraphy was performed in 61 patients at 3 months, and all patients were followed up clinically for 23 ± 15 months.

Utilizing predetermined radionuclide angiographic criteria, 47 patients (Group I) had normal right ventricular function, 12 patients (Group II) had mild to moderate dysfunction and 15 patients (Group III) had severe right ventricular dysfunction. There were no significant differences among the groups with regard to age, history of prior myocardial infarction, peak creatine kinase values, maximal Killip functional class, number or type of in-hospital complications, left ventricular ejection fraction, prevalence of multivessel disease or the distribution and severity of disease affecting the infarct-related vessel. Exercise tolerance as assessed by treadmill time, blood pressure-heart rate product and peak work load in METS was comparable among the three groups, both before hospital discharge and at 3 month follow-up. No differences in indicators of exercise-induced ischemia were noted among the groups, including the prevalence of redistribution thallium-201 defects, ST segment depression or symptoms of chest pain. Finally, cardiac mortality, reinfarction rate and the incidence of medically refractory angina pectoris were similar in the three groups. Thus, right ventricular dysfunction after acute inferior wall myocardial infarction does not appear to limit exercise tolerance or identify a subgroup of patients at higher risk for recurrent cardiac events.

(J Am Coll Cardiol 1985;6:995–1003)

Since 1973 when Cohn et al. (1) first described the clinical syndrome of right ventricular infarction, several investigators (2–11) have reported new information regarding this entity. This has stimulated renewed interest and led to greater insight concerning the status and clinical implications of right ventricular function during acute inferior wall infarction. Although a great deal is now known about the hemodynamic impact of right ventricular dysfunction in the acute setting, few data exist that address the subsequent effect on exercise performance or the risk of future cardiac events.

Accordingly, the purpose of the present study was to prospectively evaluate patients with acute inferior wall myocardial infarction to: 1) determine the prevalence of right ventricular dysfunction as assessed by predischarge gated blood pool scintigraphy, and 2) compare the extent of angiographic coronary obstructive disease, amount of left ventricular dysfunction, incidence of in-hospital complications and postdischarge events and results of serial exercise tolerance testing and quantitative thallium-201 scintigraphy in patients with and without right ventricular dysfunction. On the basis of previous studies (12–14), we hypothesized that exercise performance and risk of recurrent cardiac events would be comparable in these two groups of patients if the extent and distribution of underlying coronary artery disease, left ventricular ejection fraction values and prevalence of inducible ischemia were similar.

From the Division of Cardiology, Department of Internal Medicine and Division of Nuclear Medicine, Department of Radiology, University of Virginia Medical Center, Charlottesville, Virginia. This study was supported in part by a grant-in-aid from the American Heart Association, Virginia Affiliate, Richmond, Virginia. Manuscript received February 26, 1985; revised manuscript received June 11, 1985, accepted July 12, 1985.

Address for reprints: Robert S. Gibson, MD, Director, Cardiac Noninvasive Laboratories, University of Virginia Medical Center, Division of Cardiology, Box 468, Charlottesville, Virginia 22908.

0735-1097/85/$3.30

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Methods

Study patients. The study group comprised 74 consecutive patients with acute inferior wall myocardial infarction who were part of an ongoing prospective natural history study of survivors of uncomplicated myocardial infarction. This cohort was selected from 131 patients admitted to our coronary intensive care unit who met the following criteria: 1) acute myocardial infarction diagnosed by typical history of chest pain and a diagnostic rise and fall of the serum creatine kinase-MB isoenzyme; 2) age 65 years old or younger; 3) absence of significant valvular, congenital or cardiomyopathic heart disease or history of coronary bypass surgery; 4) absence of cardiogenic shock, ventricular septal defect or papillary muscle rupture; 5) absence of serious noncoronary disease, including significant chronic obstructive pulmonary disease, that might limit or confound long-term follow-up study; and 6) willingness to give informed consent and undergo graded exercise testing, quantitative thallium-201 scintigraphy, multigated blood pool scintigraphy and coronary angiography before hospital discharge. In addition, patients were asked to return to the postmyocardial infarction clinic at specified intervals for follow-up study. These study patients were a representative subgroup of all patients at our institution with uncomplicated infarction and were similar to eligible patients refusing enrollment except for slightly younger age and lower proportion of women as previously described (12).

All 74 patients met standard electrocardiographic criteria for inferior (15) or true posterior wall infarction of the left ventricle (16). Criteria used included: 1) the appearance of new Q waves 30 ms or longer in two of the three inferior leads (II, III or aVF); 2) new high amplitude R waves in leads V_1 and V_2 with an R/S ratio of 1 or greater; or 3) active evolution of ST and T wave changes in leads II, III or aVF without subsequent Q wave development.

Clinical evaluation. All patients were evaluated on admission and daily thereafter by a staff cardiologist and a research nurse for the duration of their hospitalization. Each patient was assigned to Killip functional class I to III by established criteria (17). Serum creatine kinase and MB fraction levels were determined on admission and every 4 hours thereafter until peak concentration was established, and then daily until a normal value was attained. Electrocardiograms were performed on admission and daily thereafter for 3 days. In instances of suspected infarct extension or rest angina, repeat electrocardiograms during pain were obtained.

Postmyocardial infarction complications were screened for, evaluated and recorded, including angina pectoris, infarct extension, congestive heart failure, hypotension, ventricular tachycardia or fibrillation, heart block requiring pacemaker insertion, pericarditis or right ventricular infarction syndrome. The latter was defined as hypotension and central venous pressure elevation greater than pulmonary capillary wedge pressure, or significant jugular venous distension without corresponding evidence of left heart failure (1).

Predischarge exercise testing. An intravenous cannula was inserted before the test and baseline electrocardiograms were recorded with the patient in supine, sitting and standing positions, and after 30 seconds of hyperventilation. All patients were exercised on a treadmill using the Naughton protocol (18) (n = 70) or the Bruce protocol (19) (n = 4) at a mean of 11 ± 4 days after the onset of acute infarction. Before the exercise test, no attempt was made to alter medical therapy which was to be continued chronically. Exercise was discontinued at a peak rate of 120 beats/min or 5 metabolic equivalents (METS), or if limiting symptoms or signs developed, including angina pectoris, dyspnea, fatigue, frequent (>10/min), multifocal or paired ventricular extrasystoles, ST segment depression of 4 mm or more or a decrease in systolic blood pressure of 10 mm Hg or greater below the peak blood pressure during the protocol. An intravenous dose of 1.5 to 2.0 mCi of thallium-201 was administered, followed by a 10 ml saline solution flush as the patient approached either the target heart rate/work load or limiting symptoms, and the exercise was continued as tolerated for an additional 60 seconds.

All patients were monitored continuously throughout exercise and recovery, and 12 lead electrocardiograms were recorded at 1 minute intervals during exercise and at 1, 2, 3 and 5 minutes during recovery. Heart rate and blood pressure were measured at 1 minute intervals during exercise, as were peak heart rate, blood pressure, rate-pressure product, work load and total exercise time. All tests were examined by two independent experienced observers without knowledge of patient identity or results of other studies. In cases of discordant interpretation, a consensus reading with a third observer was used.

Three month exercise testing. A similar exercise test protocol was employed in 61 patients 3 months after the onset of acute myocardial infarction using the Naughton (n = 7) or the Bruce (n = 54) protocol, with the exception that all tests were targeted either to limiting signs or symptoms or maximal age-predicted heart rate. Thirteen patients did not return for the 3 month exercise test, either because of death (n = 3), coronary artery bypass surgery or percutaneous transluminal angioplasty (n = 6), unstable angina (n = 1) or refusal to undergo repeat testing (n = 3).

Quantitative thallium-201 scintigraphy. Thallium imaging commenced 10 minutes after isotope injection. Images were obtained in the anterior and 45° and 70° left anterior oblique projections using a gamma camera with an all purpose collimator and a 25% window centered on the 80 KeV peak. Similar projections were acquired at 1 hour and 2 to 3 hours after injection. Standardized image formation and quantification of relative thallium-201 activity in seven stan-

dard myocardial scan segments were performed by methods previously described (12). Segmental thallium-201 uptake and washout were graded, as well as presence or absence of increased lung uptake and estimate of number of significantly diseased coronary vessels. All studies were interpreted by two independent experienced observers who were unaware of the clinical information. In cases of discordant interpretation, a consensus reading with a third observer was used.

Multigated blood pool imaging. After the delayed thallium-201 images were obtained, equilibrium gated blood pool imaging was performed at rest to determine left ventricular ejection fraction, right ventricular function and segmental wall motion patterns. Patients received an injection of unlabeled stannous pyrophosphate, followed 30 minutes later by 20 mCi of technetium-99m pertechnetate to complete the in vivo labeling of red blood cells. After equilibration of the blood pool tracer, imaging commenced in the anterior projection, followed sequentially by the 45° left anterior oblique (angle was modified if necessary to achieve best ventricular separation) and 70° left anterior oblique views. A 15 inch (38 cm) gamma camera was used equipped with a parallel hole collimator using a 20% energy window. The images were collected over 8 minutes using a 2 × image magnification with the computer gated by an electrocardiographic R wave detector. The cardiac cycle was divided into 16 equal segments, and counts were collected in each frame over 8 minutes.

Left ventricular ejection fraction was calculated from the 45° left anterior oblique (or modified best septal) projection without caudal angulation using a standard volume count method.

For analysis of regional wall motion, scintigraphic data were displayed in an endless loop movie format. Wall motion was assessed qualitatively by dividing the left ventricle into 11 segments using the anterior, 45° and 70° left anterior oblique projections (Fig. 1) (20). Each segment was evaluated for the presence and degree of asynergy according to the terminology used by Herman and Gorlin (21). Left and right ventricular wall motion was graded by three experienced observers without knowledge of rest or exercise electrocardiographic, thallium-201 or angiographic findings, and a consensus interpretation was obtained based on a 5 point scale: −1 = hyperkinetic; 0 = not seen; 1 = normal; 2 = hypokinetic; 3 = akinetic and 4 = dyskinetic. A regional wall motion score was derived by summing the scores of individual segments. The wall motion index was then calculated as the total wall motion score divided by the number of segments analyzed.

Global right ventricular function was designated normal (Group I), mild to moderately depressed (Group II) or severely depressed (Group III) by a blinded consensus reading from the three experienced observers. The research reading was compared with the clinical reading and, if discordant, an arbitration reading by a fourth experienced observer was solicited. Because of the complex and variable geometry of the right ventricle, as well as the well known difficulties with right atrial separation (20,22–25), no attempt was made to numerically describe the global right ventricular ejection fraction. A wall motion score was derived as described using a visual average of free wall motion. In addition, right ventricular size was scored as: 1 = normal, 2 = mild enlargement and 3 = severe enlargement.

Coronary angiography. Selective coronary angiography using either the Judkins (26) or Sones (27) technique was performed in all patients within 24 to 72 hours of exercise testing and gated blood pool imaging. Coronary angiograms were reviewed by two experienced angiographers without knowledge of electrocardiographic or radionuclide findings. The maximal luminal diameter narrowing for each major coronary artery (right, left anterior descending and circumflex) was visually estimated and considered hemodynamically significant if it was 50% or greater in any projection. The location of each recognized narrowing was defined according to the 15 segment model recommended by the American Heart Association (28). Only the most severe narrowing of the coronary artery segment was recorded and each patient classified as having one, two or three vessel disease. Significant narrowing in large diagonal or marginal branches were considered lesions of the left

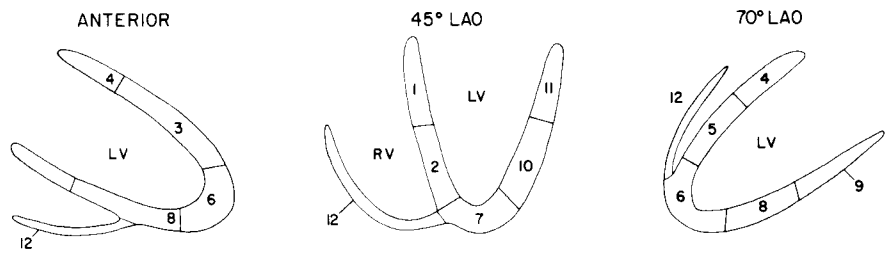

Figure 1. Model of radionuclide ventriculogram employing 11 left ventricular segments and 1 right ventricular segment used to grade regional left ventricular and right ventricular free wall motion by equilibrium gated blood pool imaging. LAO = left anterior oblique. Segments: 1, basal septal; 2, apical septal; 3, anterolateral; 4, anterobasal; 5, anterior; 6, apical; 7, inferoapical; 8, inferior; 9, posterobasal; 10, inferolateral; 11, posterolateral; 12, right ventricular. Score code: −1, hyperdynamic; 0, not seen; +1, normal; +2, hypokinetic; +3, akinetic; +4, dyskinetic.

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anterior descending or left circumflex artery, respectively. Narrowing of the left main coronary artery was recorded as disease in both the left anterior descending and left circumflex arteries.

Because the study cohort was limited to patients with inferior wall infarction, only the left circumflex and right coronary arteries were considered as infarct vessels. Patients with a single vessel right coronary artery infarct usually have inferior, inferoapical and low posterolateral wall motion abnormalities, while mid and high posterolateral regions represent the risk area of the left circumflex coronary artery. Therefore, when the left circumflex and right coronary arteries were both found to be stenosed, the infarct vessel was chosen on the basis of the location of maximal asynergy by radionuclide angiocardiography. Specifically, high posterolateral wall motion abnormalities indicated a left circumflex coronary artery infarct and inferior or low posterolateral asynergy indicated a right coronary artery infarct. The infarct vessel was considered to be patent if prompt anterograde flow of angiographic dye across the vessel stenosis was demonstrated.

Follow-up. After hospital discharge, the patient was referred to the care of his or her private physician, who had access to all study results. No attempt was made to standardize therapy or regulate rehabilitation strategy. All patients were asked to return to the postmyocardial infarction clinic at 3 months, then yearly for 5 years. Repeat exercise thallium-201 scintigraphy was undertaken at 3 months, and the patient's clinical status was evaluated by one of the investigators during each visit. For those patients not returning to the clinic at the designated time, follow-up information was collected by telephone interview. The necessary information was obtained for all patients.

During the mean follow-up period of 23 ± 15 months, we specifically attempted to determine the incidence of: 1) cardiac death, 2) recurrent myocardial infarction, and 3) the development of rapidly progressive angina pectoris with minimal exertion (New York Heart Association functional class III) or angina at rest (functional class IV) of sufficient clinical concern to warrant hospitalization. Before our study began, we decided that coronary bypass surgery would not be considered an event. Because we anticipated that the results of exercise testing, thallium-201 scintigraphy or coronary angiography might contribute to the decision to perform surgery, the follow-up period was terminated in the case of surgery. The diagnosis of recurrent myocardial infarction was established as previously described. Death was considered cardiac in origin if it occurred suddenly (that is, within 1 hour of onset of symptoms) or if it was associated with other cardiac complications for which the patient had been hospitalized. For purposes of analysis, only one event, the most serious in the preceding order, was tabulated for each patient.

Statistical analysis. Individual test data were compiled prospectively and stored in a computerized data bank. Continuous data are presented as mean values ± standard deviation. To determine differences between mean values of independent observation, a one-way analysis of variance and Duncan's multiple range test were used to delineate the significance of any observed differences. Discrete variables were analyzed using contingency tables with appropriate chi-square or Fisher's exact statistics.

Results

In the group of 74 patients with inferior myocardial infarction, 47 (39 men, 8 women: Group I) had normal right ventricular function by multigated blood pool scintigraphy. A total of 27 patients (36%) had evidence of right ventricular asynergy including 12 (all men: Group II) with mild to moderate right ventricular dysfunction and 15 patients (14 men, 1 woman: Group III) with severely depressed right ventricular function (Fig. 2).

Clinical characteristics (Table 1). Table 1 depicts pertinent clinical, radionuclide and angiographic data in our three groups of patients. There were no significant differences among the groups with respect to the clinical variables evaluated, including age, history of prior myocardial infarction, peak creatine kinase value, type of acute myocardial infarction and maximal Killip class in the coronary care unit. The proportion of patients with mild to moderate (6 patients) and severe (12 patients) right ventricular dysfunction among the 51 patients with a Q wave infarction was not significantly different from that among the 23 patients with a non-Q wave infarction (6 and 3 patients, respectively).

A total of 57 in-hospital complications occurred in 44 (59%) of our 74 patients. The percent of patients within each group who experienced complications was comparable.

Figure 2. End-diastolic (**left**) and end-systolic (**right**) frames from the predischarge gated blood pool scan in a patient with severe right ventricular dysfunction. The left ventricular ejection fraction measured 60% despite obvious enlargement and significant loss of systolic function of the right ventricle.

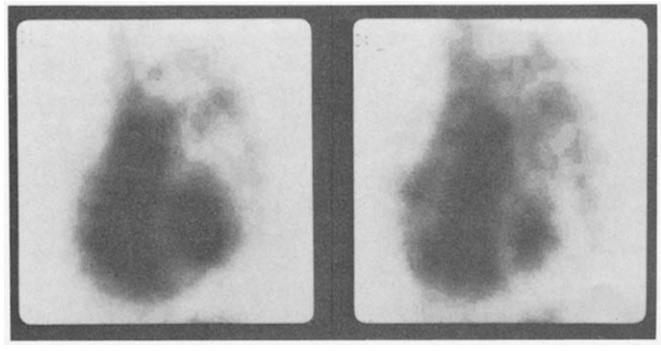


Table 1. Pertinent Clinical, Radionuclide and Angiographic Data in 74 Patients

	Group I (n = 47)	Group II (n = 12)	Group III (n = 15)	Groups II and III (n = 27)
Clinical				
Age (yr)	52 ± 7	50 ± 7	53 ± 7	52 ± 7
Prior MI	10(21%)	2(17%)	1(7%)	3(11%)
Prior anterior MI	2(4%)	0	0	0
Peak CK (IU/liter)	890 ± 967	828 ± 423	1,281 ± 821	1,080 ± 662
Type of Acute MI				
Inferior Q wave	30(64%)	6(50%)	12(80%)	18(67%)
True posterior	3(6%)	0	0	0
Inferior non-Q wave	14(30%)	6(50%)	3(20%)	9(33%)
Peak Killip class	1.6 ± 0.6	1.3 ± 0.5	1.6 ± 0.5	1.5 ± 0.5
In-hospital complications (pts)	29(62%)	6(50%)	9(60%)	15(56%)
Gated blood pool scan				
LV ejection fraction (%)	52 ± 9	54 ± 5	50 ± 8	52 ± 7
LV wall motion index	1.4 ± 0.3	1.4 ± 0.2	1.5 ± 0.2	1.5 ± 0.2
RV wall motion score	1.1 ± 0.2*	1.7 ± 0.2*	2.6 ± 0.3*	2.2 ± 0.3†
RV size index	1.0 ± 0.0‡	1.3 ± 0.3*	2.2 ± 0.3*	1.7 ± 0.3†
Angiography				
Stenosed vessels (no.)	1.8 ± 0.8	2.0 ± 0.7	2.1 ± 0.8	2.1 ± 0.8
Infarct vessels				
Right coronary artery	35(74%)	9(75%)	14(93%)	23(85%)
Left circumflex artery	12(26%)	3(25%)	1(7%)	4(15%)
Patent infarct vessel	12(26%)	4(33%)	1(7%)‖	5(19%)
Proximal infarct vessel stenosis	34(76%)	6(50%)	11(73%)	17(63%)

*$p < 0.001$ compared with other groups; †$p < 0.001$ compared with Group I; ‡$p < 0.03$ compared with Group II; ‖$p < 0.08$ compared with Groups I and II. All values are expressed as mean ± standard deviation. CK = creatine kinase; LV = left ventricular; MI = myocardial infarction; pts = number of patients; RV = right ventricular.

Of note, only four patients (5%) had a clinically recognized right ventricular infarction. Two of these patients had severe right ventricular dysfunction on predischarge gated blood pool scintigraphy and are included in Group III. The remaining two patients exhibited normal right ventricular function before discharge. This finding was corroborated by two-dimensional echocardiography.

Radionuclide and angiographic data (Table 1). The left ventricular ejection fraction was similar among the three groups, being 52 ± 9, 54 ± 5 and 50 ± 8% in Groups I to III, respectively. Also the left ventricular wall motion index was comparable among patient groups despite a wide range of values within the entire cohort.

Of 57 patients in the initial cohort of 131 patients who had acute infarction on the electrocardiogram in a distribution other than the inferior leads, only 4 (7%) showed evidence of right ventricular dysfunction on gated cardiac blood pool scanning. All but one had an obvious explanation for this right ventricular dysfunction including a history of previous inferior myocardial infarction (n = 2) and a right coronary artery occlusion as the cause of an apical infarction (n = 1) that was diagnosed as "anterior" by electrocardiographic criteria.

Finally, the angiographic variables evaluated in our three groups were similar, including the prevalence of multivessel disease. Although no appreciable difference in the occurrence of proximal infarct vessel stenosis was demonstrated, there was a trend toward fewer patent infarct vessels in Group III versus Groups I and II (7 versus 26 and 33%, respectively, p = 0.08).

Exercise performance. *Predischarge.* Exercise test results for the three groups before hospital discharge are depicted in Figure 3. Variables indicative of exercise capacity that we assessed included peak work load achieved expressed in metabolic equivalents (METS), total treadmill time and maximal rate-pressure product. In no instance were these values significantly different among Groups I to III. When Groups II and III were combined, exercise variables did not differ from the values in Group I. Although the predischarge test was targeted to a heart rate of 120 beats/min, 50 (67%) of the 74 patients failed to achieve this end point because of limiting symptoms. The proportion of patients with limiting symptoms, as well as the specific reasons for premature exercise test termination, was similar among the three groups.

Postdischarge. Since the predischarge exercise testing was submaximal by design, symptom-limited testing was employed 3 months later. Figure 4 illustrates our data from

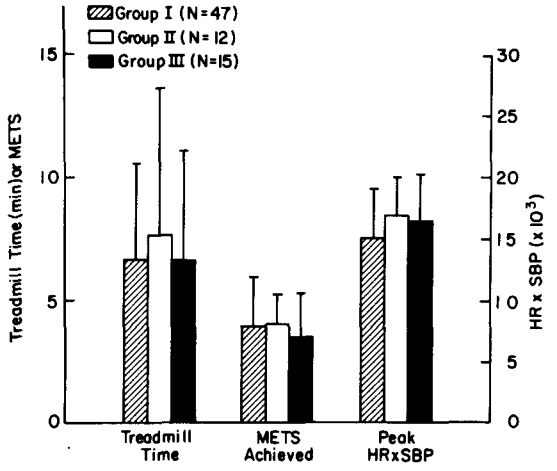
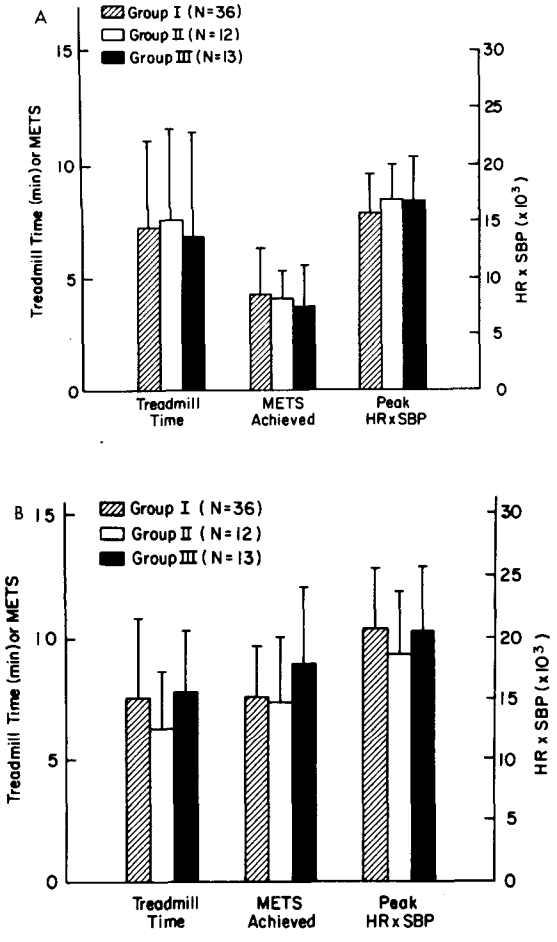


Figure 3. Exercise performance variables in 74 patients with varying right ventricular function who underwent predischarge exercise testing after acute inferior myocardial infarction (see text). HR = heart rate; SBP = systolic blood pressure.

Figure 4. Exercise performance variables in 61 patients with varying right ventricular function who underwent exercise testing both before hospital discharge (**A**) and 3 months (**B**) after acute inferior myocardial infarction (see text). HR = heart rate; SBP = systolic blood pressure.



the 61 patients who underwent testing both at 10 days and at 3 months after the onset of infarction. Again, the post-discharge studies in these patients show no significant differences among the three groups or between Group I and the combined Groups II and III with respect to exercise performance. As expected, the 3 month data showed an overall increase in exercise capacity; however, the increment in each of the analyzed variables was comparable among the three groups.

Quantitative thallium-201 scintigraphy (Table 2). Compared with Group I, patients in Groups II and III were similar with regard to the number of left ventricular segments demonstrating abnormal uptake or washout of thallium-201, or both, the prevalence of thallium defects involving more than one discrete vascular region (multivessel disease pattern) and the number of segments showing redistribution or a persistent defect. Also, the prevalence of increased lung uptake of thallium on the initial unprocessed scintiphotographs was similar in the three groups. Thus, the extent of residual ischemia, magnitude of scar and evidence of exercise-induced left ventricular dysfunction appeared comparable among Groups I to III.

Clinical follow-up (Table 3). During a mean follow-up period of 23 ± 15 months (range 1 to 60), 6 patients (8%) died, 5 (7%) had another infarction and 10 (13.5%) experienced unstable angina pectoris. Sixteen (22%) underwent coronary bypass surgery or percutaneous transluminal angioplasty because of progressive angina pectoris refractory to medical therapy (4 patients) or because of unstable angina pectoris (12 patients, 1 of whom also had significant left main coronary artery occlusion). Overall, 25 patients had one or more of these events.

Because the prevalence of exercise-induced ST segment depression, angina pectoris and thallium-201 scintigraphic markers of high risk were similar in the three groups, it is not surprising that the incidence of individual or combined cardiac events was comparable among Groups I to III. Overall, 63% of the patients were able to return full-time to their previous occupation.

Discussion

Our results demonstrate that right ventricular dysfunction as assessed by gated blood pool imaging 11 days after the onset of acute inferior myocardial infarction is common, occurring in 36% of our study patients. However, its presence does not appear to effect exercise performance or the incidence of subsequent cardiac events.

Clinical features. Previous reports have described a wide range in the incidence of right ventricular dysfunction during acute inferior myocardial infarction, depending on patient selection, the criteria used to define right ventricular infarction and the time after onset of myocardial infarction when evidence of right ventricular involvement was sought.

Table 2. Thallium-201 Results at Discharge in 74 Patients

Thallium-201 Scintigraphic Variable	Group I (n = 47)	Group II (n = 12)	Group III (n = 15)
Abnormal LV segments (no.)	2.4 ± 0.1	2.3 ± 0.3	2.5 ± 0.3
Multivessel disease pattern (pts)	22(47%)	5(42%)	7(47%)
LV segments with redistribution (no.)	0.8 ± 0.2	0.9 ± 0.3	0.9 ± 0.3
LV segments with severe persistent defects†	1.6 ± 0.1	1.3 ± 0.3	1.7 ± 0.2
Increased lung uptake (pts)	11(24%)	1(8%)*	4(27%)

*p = NS. All values are expressed as mean ± standard deviation. † > 50% diminished thallium-201 uptake. Abbreviations as in Table 1.

For example, a recent review (29) of 12 postmortem studies comprising 1,997 patients indicated that between 1 and 43% show right ventricular necrosis. When radionuclide or echocardiographic techniques are employed before hospital discharge, the reported incidence has ranged from 27 to 50% (5–11). Thus, the 36% incidence of right ventricular dysfunction in our study patients at 11 days is not unexpected.

Our study identified no significant premorbid clinical predictors of right ventricular dysfunction, including history of previous myocardial infarction or extent of left ventricular involvement as determined by left ventricular ejection fraction values and wall motion indexes. Although not statistically significant, creatine kinase values were higher in the group with the most severe right ventricular dysfunction and probably represented enzyme release from infarcting right ventricular myocardium (30).

Clinical evidence of right ventricular dysfunction was found in only 7% of our patients in Groups II and III. This finding is not surprising since previous observations (1,2) indicate that clinical criteria for the diagnosis of right ventricular infarction are relatively insensitive. Of interest were the two patients who were thought to have clinical right ventricular dysfunction acutely, but who demonstrated normal right ventricular function before hospital discharge. Either the right ventricular dysfunction resolved over the 11 day convalescent period or these cases represented false positive clinical diagnoses.

Exercise performance. It has been proposed that the presence and magnitude of inducible ischemia and overall left ventricular functional reserve represent major determinants of exercise performance in patients with known coronary artery disease (31). Ischemia may contribute to decreased exercise tolerance by causing limiting anginal symptoms (32,33) or by causing ischemic left ventricular dysfunction that might lead to significant dyspnea or fatigue (34–37). Investigators (14) have also determined that, in general, left ventricular function at rest and contractile reserve correlate with exertional capacity. Additionally, peripheral factors and training effects will modulate an individual patient's exertional tolerance (38,39). The three groups in our study were comparable with respect to the angiographic extent of coronary artery disease, prevalence of ST segment depression or angina, or both, number of segments with thallium-201 redistribution and global and regional left ventricular function at rest. Therefore, the observation of similar exercise performance among groups was not unexpected.

Our data apply only to a group of patients who were capable of undergoing predischarge exercise testing. As such, their postmyocardial infarction convalescence was relatively benign and all met criteria for an uncomplicated acute myocardial infarction by day 5 (40). Also, only two patients with right ventricular dysfunction on the predischarge radionuclide study had signs of clinical right ventricular infarction

Table 3. Clinical Outcome in 74 Patients With Inferior Wall Infarction

	Group I (n = 47)	Group II (n = 12)	Group III (n = 15)
Duration of follow-up (mo)	23 ± 17	24 ± 8	24 ± 14
Cardiac death	3(6%)	2(17%)	1(7%)
Reinfarction	3(6%)	1(8%)	1(7%)
Unstable angina	7(15%)	1(8%)	2(13%)
Myocardial revascularization*	12(26%)	2(17%)	2(13%)
Returned to work	27(57%)	8(67%)	12(80%)

*By coronary bypass surgery or percutaneous transluminal angioplasty.

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acutely. Perhaps, a larger group of patients that included more patients with clinical right ventricular failure would show a trend toward impaired exercise performance.

It has been suggested that right-sided stroke volume is highly dependent on ventricular septal function, particularly when the free wall of the right ventricle has been damaged (41–45). Since only two patients (3%) in the present cohort had a history of prior anteroseptal myocardial infarction and only one had evidence of major myocardial asynergy involving the septum, it was not possible for us to evaluate septal function as a factor affecting exercise performance in the setting of right ventricular dysfunction. Further studies to assess the influences of interventricular septal function in right ventricular infarction would be of value.

Clinical outcome. The cardiac event rates observed in our 74 patients were relatively low, but were anticipated because postmyocardial infarction patients who are able to undergo exercise testing 10 to 14 days after the onset of infarction represent, in general, a low risk subgroup. In agreement with our original hypothesis, we found similar cardiac event rates among patients with normal and impaired right ventricular function (Table 3), which suggests that factors other than right ventricular dysfunction have a greater influence on prognosis.

To our knowledge, no study has yet established right ventricular dysfunction as an independent predictor of adverse outcome after recovery from an otherwise uncomplicated acute myocardial infarction. However, many groups have demonstrated that decreased left ventricular function at rest, impaired exercise tolerance, complex ventricular ectopic activity and stress-induced ischemia are all related to poor prognosis. Because our three patient groups were similar with regard to these prognostic variables, it is not surprising that comparable cardiac event rates were observed.

Clinical significance. The interest in recent years in identifying patients with acute right ventricular infarction syndrome has led to the detection of a large number of patients with significant right ventricular dysfunction diagnosed by noninvasive techniques, most of whom apparently have an unremarkable clinical course. The results of the present study, however, suggest that overall cardiac reserve capacity during exercise is not limited by impaired right ventricular function, and long-term outcome is likewise unaffected. Management of the individual patient after recovery from inferior myocardial infarction is more likely dependent on the degree of left ventricular dysfunction and the presence and extent of residual ischemia. Although extensive right ventricular infarction can have a profound hemodynamic impact in the acute setting, the long-term effects in patients such as those represented in our cohort appear benign.

We thank Elizabeth Howk for her excellent secretarial assistance.

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